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Original Research Article

Unexpected red cell antibodies in pregnancy and their obstetric implications Beyond RhD: The Hidden Burden of Unexpected Maternal Red Cell Antibodies and Their Perinatal Consequences

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Conflict of interest: Nil

Abstract:

Background: Unexpected maternal red cell antibodies can cause fetal anaemia, haemolytic disease of the fetus and newborn, neonatal jaundice, and perinatal loss. Early detection is essential for surveillance and transfusion planning.

Methods: This prospective study was conducted at Kakatiya Medical College and CKM Hospital, Warangal from November 2025 to July 2026. Five hundred women underwent ABO and RhD typing and antibody screening by indirect antiglobulin testing. Positive samples were evaluated for antibody specificity. Maternal risk factors, obstetric history, fetal complications, and neonatal outcomes were recorded and analysed.

Results: Unexpected red cell antibodies were detected in 18 women, giving a prevalence of 3.6%. Anti-D was most common (38.9%), followed by anti-c and anti-E (16.7% each), anti-K and anti-M (11.1% each), and combined anti-D with anti-C (5.6%). Antibody positivity was significantly associated with multigravidity, previous abortion, stillbirth or intrauterine fetal death, prior blood transfusion, RhD-negative status, and maternal age 30 years or older. Antibody-positive pregnancies had higher rates of preterm birth, fetal anaemia, intrauterine transfusion, low birth weight, neonatal jaundice, positive direct antiglobulin test, exchange transfusion, NICU admission, and perinatal mortality.

Conclusion: Unexpected red cell antibodies were associated with adverse obstetric and neonatal outcomes. Antenatal antibody screening and multidisciplinary management are warranted.

Keywords: Alloimmunization; Antibody screening; Fetal anaemia; Haemolytic disease of the fetus and newborn; Pregnancy.

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Introduction

Unexpected red cell antibodies arise when a pregnant woman becomes immunized against erythrocyte antigens inherited by the fetus, usually after fetomaternal haemorrhage, previous pregnancy, abortion, invasive procedures or blood transfusion [1]. Although anti-D remains the best-recognized antibody, clinically significant non-D antibodies such as anti-c, anti-E, anti-K and anti-M may cause fetal anaemia, hydrops, intrauterine death, neonatal hyperbilirubinaemia and the need for intrauterine or exchange transfusion. Indian evidence indicates that alloimmunization is frequent among RhD-negative women, multigravidae and women with adverse obstetric histories, while antibodies may also occur in RhD-positive pregnancies [2, 3]. Recent Indian studies have highlighted variation in antibody prevalence, inadequate universal screening and the importance of early identification, antibody characterization and coordinated obstetric–transfusion support [3]. Because affected pregnancies may remain clinically silent until fetal compromise develops, routine antenatal antibody screening can facilitate titre monitoring, fetal middle cerebral artery Doppler surveillance and appropriate intervention. The present study aimed to determine the frequency and specificity of unexpected red cell antibodies in pregnant women and evaluate their associations with maternal characteristics, obstetric history, fetal

complications, neonatal morbidity and perinatal outcome.

Methods

A prospective observational study was conducted in the Department of Obstetrics and Gynaecology in collaboration with the Department of Transfusion Medicine at Kakatiya Medical College and CKM Hospital, Warangal, from November 2025 to July 2026. Pregnant women attending the antenatal outpatient department or admitted to the obstetric wards during the study period were screened for eligibility. All consecutive pregnant women, irrespective of gestational age, gravida, parity or ABO and RhD blood group, who provided written informed consent were included. Women who had received immunoglobulin therapy recently, had autoimmune haemolytic anaemia, had undergone organ transplantation or had incomplete clinical records were excluded. Ethical clearance was obtained from the Institutional Ethics Committee before the commencement of the study. A structured case-record form was used to document age, residence, socioeconomic status, gestational age, gravida, parity, previous abortions, stillbirths, intrauterine deaths, neonatal deaths, history of haemolytic disease of the fetus and newborn, previous blood transfusions, invasive obstetric procedures and administration of anti-D immunoglobulin. Details of the current pregnancy, including maternal medical disorders, obstetric complications and ultrasonographic findings, were also recorded.

A venous blood sample of approximately 5 mL was collected aseptically from each participant into an ethylenediaminetetraacetic acid tube. ABO and RhD typing was performed using standard serological techniques with commercially available monoclonal antisera. Antibody screening was carried out using a validated three-cell screening panel by the indirect antiglobulin test, either through column agglutination technology or a conventional tube method according to the laboratory protocol. Samples showing a positive antibody screen were further evaluated using an extended red cell identification panel to determine antibody specificity. Autocontrol and direct antiglobulin testing were performed when required to differentiate alloantibodies from autoantibodies. Clinically significant antibodies were defined as antibodies reactive at 37°C or in the antihuman globulin phase and known to be associated with haemolytic disease of the fetus and newborn or haemolytic transfusion reactions. Antibody titration was performed in women with clinically significant antibodies, and titres were repeated according to obstetric and transfusion-medicine recommendations. Paternal antigen testing and fetal antigen assessment were undertaken where clinically indicated and feasible. Women with clinically significant antibodies were managed jointly by obstetricians, fetal-medicine specialists, neonatologists and transfusion-medicine personnel.

Pregnancies with detected unexpected red cell antibodies were followed prospectively until delivery. Maternal monitoring included serial antibody titres, haemoglobin estimation and assessment for obstetric complications. Fetal surveillance included ultrasonography for growth, liquor volume, placental changes and features of hydrops. Middle cerebral artery peak systolic velocity Doppler was performed when fetal anaemia was suspected. The need for intrauterine transfusion, timing and mode of delivery and availability of antigen-negative compatible blood were recorded. Neonatal outcomes included birth weight, gestational age at delivery, Apgar scores, cord haemoglobin, bilirubin concentration, direct antiglobulin test result, neonatal anaemia, jaundice, phototherapy, intravenous immunoglobulin, exchange transfusion, neonatal intensive care admission and perinatal mortality. Data were entered into Microsoft Excel and analysed using appropriate statistical software. Continuous variables were expressed as mean and standard deviation or median and interquartile range, while categorical variables were presented as frequencies and percentages. Associations between antibody positivity and maternal, obstetric and neonatal variables were assessed using the chi-square test, Fisher's exact test, independent-samples t-test or Mann–Whitney U test, as appropriate. A p-value below 0.05 was considered statistically significant.

Results

The study included 500 pregnant women, of whom 18 had a positive antibody screen, giving an overall unexpected red cell antibody prevalence of 3.6%. As shown in Table 1, antibody-positive women were significantly older than antibody-negative women (28.7 ± 4.6 versus 25.8 ± 4.1 years; p=0.004). Positivity was significantly more frequent among multigravidae, women with previous abortions, stillbirth or intrauterine fetal death, previous blood transfusion and RhD-negative blood groups. The mean gestational age at screening did not differ significantly between the groups. Anti-D was the most frequently identified antibody, accounting for 38.9% of positive samples, followed by anti-c and anti-E at 16.7% each. Anti-K, anti-M and combined anti-D with anti-C were less frequent (Table 2). Risk-factor analysis showed that RhD-negative women had approximately 15.6-fold greater odds of antibody positivity, while previous blood transfusion was associated with nearly 11-fold higher odds. Previous stillbirth or intrauterine fetal death, multigravidity and maternal age of at least 30 years were also significantly associated with alloimmunization (Table 3). Adverse obstetric and neonatal outcomes occurred more frequently in

antibody-positive pregnancies. These women had significantly higher rates of preterm delivery, fetal anaemia, low birth weight and intrauterine transfusion. Their neonates more frequently developed a positive direct antiglobulin test, significant jaundice requiring phototherapy, exchange transfusion and neonatal intensive care admission. Perinatal mortality was 11.1% in antibody-positive pregnancies compared with 1.0% among antibody-negative pregnancies (p=0.008), demonstrating the clinically important obstetric implications of unexpected red cell antibodies (Table 4).

Table 1: Baseline maternal and obstetric characteristics according to antibody-screening status

Characteristic	Antibody screening result		Total	p-value
	Negative	Positive		
Maternal age, years*	25.8 ± 4.1	28.7 ± 4.6	25.9 ± 4.2	0.004
Gestational age**	18.9 ± 7.2	20.1 ± 7.6	19.0 ± 7.2	0.486
Multigravida	259 (53.7%)	15 (83.3%)	274 (54.8%)	0.014
Previous abortion	89 (18.5%)	7 (38.9%)	96 (19.2%)	0.032
Previous stillbirth/IUFD	17 (3.5%)	4 (22.2%)	21 (4.2%)	<0.001
Previous blood transfusion	21 (4.4%)	6 (33.3%)	27 (5.4%)	<0.001
RhD-negative blood group	38 (7.9%)	10 (55.6%)	48 (9.6%)	<0.001
*mean ± SD; **Gestational age at screening, weeks, mean ± SD' IUFD: intrauterine fetal death				

Table 2: Frequency and specificity of unexpected red cell antibodies

Antibody specificity	Number (%)	% among all women
Anti-D	7 (38.9)	1.4
Anti-c	3 (16.7)	0.6
Anti-E	3 (16.7)	0.6
Anti-K	2 (11.1)	0.4
Anti-M	2 (11.1)	0.4
Anti-D with anti-C	1 (5.6)	0.2
Total	18 (100)	3.6

Table 3: Factors associated with unexpected red cell antibody positivity

Risk factor	Antibody-positive/total	Positivity rate	Unadjusted OR (95% CI)	p-value
Multigravida	15/274	5.5%	4.64 (1.32–16.29)	0.01
Previous abortion	7/96	7.3%	3.03 (1.14–8.07)	0.021
Previous stillbirth/IUFD	4/21	19%	7.87 (2.35–26.36)	<0.001
Previous blood transfusion	6/27	22.2%	10.95 (3.76–31.91)	<0.001
RhD-negative blood group	10/48	20.8%	15.58 (5.77–42.08)	<0.001
Maternal age ≥30 years	8/111	7.2%	2.94 (1.13–7.67)	0.022

Table 4: Obstetric and neonatal outcomes according to antibody status

Outcome	Antibody screening result		p-value
	Negative	Positive	
Preterm delivery	43 (8.9%)	5 (27.8%)	0.018
Fetal anaemia	2 (0.4%)	4 (22.2%)	<0.001
Intrauterine transfusion	0	2 (11.1%)	<0.001
Low birth weight	61 (12.7%)	7 (38.9%)	0.001
Neonates requiring phototherapy	36 (7.5%)	8 (44.4%)	<0.001
Positive*	4 (0.8%)	9 (50.0%)	<0.001
Exchange transfusion	1 (0.2%)	2 (11.1%)	<0.001
NICU admission	42 (8.7%)	8 (44.4%)	<0.001
Perinatal mortality	5 (1.0%)	2 (11.1%)	0.008
* neonatal direct antiglobulin test			

Discussion

The present prospective study demonstrated an overall unexpected red cell antibody prevalence of 3.6% among 500 pregnant women. This finding confirmed that maternal red cell alloimmunization remained an important clinical problem despite the

availability of RhD immunoprophylaxis. The prevalence observed in this study was comparable with the 3.12% alloimmunization rate reported by Gothwal et al. among antenatal women at a tertiary centre in Western India, but it was higher than the 2.27% reported by Das et al. in a prospective Indian study [1, 4]. Golia et al. identified unexpected antibodies in approximately 1% of antenatal women, whereas studies from Gujarat, West Bengal and South India reported variable frequencies depending on the population screened, inclusion of RhD-positive women, obstetric risk profile and laboratory method employed [5, 6]. A recent Indian systematic review and meta-analysis also demonstrated considerable heterogeneity in the reported prevalence of antenatal alloimmunization, reflecting regional differences in RhD-negative frequency, transfusion practices, anti-D coverage and access to sensitive antibody-screening facilities [3]. The relatively higher prevalence in the present study might have resulted from the tertiary-care setting, where women with previous adverse pregnancies and suspected high-risk conditions were more likely to attend. The use of a three-cell screening panel and antiglobulin-phase testing may also have improved the detection of weak or uncommon antibodies. These observations supported universal antibody screening rather than limiting testing to RhD-negative women, because clinically significant non-D antibodies may occur in women of any ABO or RhD group.

Antibody-positive women in the present study were significantly older than antibody-negative women, and alloimmunization was more frequent among multigravidae. Women with previous abortion, stillbirth, intrauterine fetal death or blood transfusion also had significantly higher antibody positivity. These findings were biologically plausible because each pregnancy, delivery, abortion, invasive procedure or transfusion may expose an antigen-negative woman to foreign red cell antigens and stimulate an immune response. Multigravidae had approximately 4.6-fold greater odds of alloimmunization, while a history of stillbirth or intrauterine fetal death was associated with almost eightfold greater odds. Studies from India have similarly found that gravida, parity, previous transfusion and inadequate anti-D prophylaxis were important determinants of maternal alloimmunization [7, 8]. Dholakiya et al. reported a high rate of alloimmunization among RhD-negative multiparous women and emphasized strict adherence to immunoprophylaxis [6]. Naik et al. identified significant associations between antenatal alloimmunization and previous obstetric exposure in women from West Bengal [7]. An Indian genetic study further showed that the frequency of anti-D alloimmunization was associated with gravida and previous anti-D administration, while also suggesting that maternal HLA characteristics might influence individual susceptibility [8]. The association with advanced maternal age probably reflected cumulative exposure rather than age acting independently. Older pregnant women were more likely to have experienced multiple pregnancies, abortions, operative deliveries or transfusions. Therefore, a detailed obstetric and transfusion history remained essential, although the absence of an identifiable exposure should not be used to exclude antibody screening.

RhD-negative status emerged as the strongest risk factor in the present study, with antibody positivity occurring in 20.8% of RhD-negative women compared with a much lower proportion among RhD-positive women. RhD-negative women had approximately 15.6-fold higher odds of possessing unexpected antibodies. Anti-D was the most common specificity and accounted for 38.9% of detected antibodies, while one woman had combined anti-D and anti-C. This distribution was consistent with several Indian reports in which anti-D remained the leading clinically significant antibody [1,3,4,6]. Das et al. found anti-D in nearly half of alloimmunized pregnancies and also identified anti-D with anti-C, anti-G and other Rh antibodies [4]. Gothwal et al. similarly observed anti-D as the dominant specificity, with combinations involving anti-C or anti-G in some women [1]. The persistence of immune anti-D in India may indicate missed antenatal prophylaxis, failure to administer postnatal anti-D after an RhD-positive birth, inadequate dosing after large fetomaternal haemorrhage, failure to provide anti-D after abortion or invasive procedures, or delayed antenatal registration. Recombinant anti-D has been shown in an Indian multicentre clinical trial to have efficacy and safety comparable with conventional polyclonal anti-D, suggesting a potential means of improving access to prophylaxis [9]. However, prophylaxis prevents only anti-D formation and has no effect on anti-c, anti-E, anti-K, anti-M or other clinically significant antibodies. The distinction between passive anti-D after prophylaxis and true immune anti-D was also important, because misclassification could lead either to unnecessary intensive surveillance or failure to recognize a genuinely sensitized pregnancy.

Non-D antibodies constituted more than half of all antibodies identified in the present study. Anti-c and anti-E each accounted for 16.7%, while anti-K and anti-M each accounted for 11.1%. This finding had important implications because a screening strategy restricted to RhD typing would have failed to identify many at-risk pregnancies. Anti-c can cause severe fetal anaemia comparable with that produced by anti-D, while anti-E may cause variable disease and may increase severity when present with anti-c. Anti-K differs mechanistically from many Rh

antibodies because it can suppress fetal erythropoiesis in addition to causing red cell destruction; consequently, severe fetal anaemia may occur even when maternal titres are not markedly elevated. Anti-M is often naturally occurring and predominantly immunoglobulin M, but its immunoglobulin G component can cross the placenta and occasionally produce severe fetal anaemia, neonatal jaundice or prolonged neonatal anaemia. Indian case reports and series have documented clinically important disease due to anti-E, anti-S, anti-M, Kidd antibodies and combinations of Rh and MNS-system antibodies [10, 11]. Pahuja et al. described dual maternal alloimmunization in which one antibody masked another, demonstrating the limitations of interpreting reactions without a complete identification panel [10]. Gupta et al. and Varghese et al. documented haemolytic disease caused by multiple antibodies, particularly in women with previous pregnancies or repeated transfusions [11, 12]. The present antibody profile therefore supported screening all pregnant women at booking and repeating testing later in pregnancy when clinically indicated, even when the initial screen was negative, because antibodies such as anti-E may become detectable after renewed antigen exposure [13].

Previous blood transfusion was one of the most powerful predictors in this study. Antibody positivity was observed in 22.2% of previously transfused women, and these women had almost 11-fold increased odds of alloimmunization. Transfusion exposes recipients simultaneously to numerous donor red cell antigens and can produce antibodies that remain detectable for years or decline below routine detection before showing an anamnestic response during pregnancy. This problem is particularly relevant when girls and women of reproductive age receive emergency transfusions, transfusions for anaemia, obstetric haemorrhage, haemoglobinopathies or surgery without extended antigen matching. The identification of anti-K, anti-E, anti-c and other antibodies among pregnant women may therefore reflect previous transfusion as well as fetomaternal exposure. Indian transfusion studies have consistently recommended antibody screening and improved antigen matching in repeatedly transfused and previously pregnant individuals [14]. Partial matching for Rh and Kell antigens has been proposed as a practical strategy to reduce alloimmunization in susceptible Indian patients [15]. For women of childbearing potential, avoiding Kell-positive blood for Kell-negative recipients and providing Rh phenotype-compatible units whenever feasible could reduce future HDFN. Once an antibody was identified, its specificity needed to be permanently documented because it might become serologically undetectable while remaining clinically relevant. Antigen-negative, crossmatch-compatible blood should be reserved for the mother, and compatible group O, antigen-negative, cytomegalovirus-safe and appropriately processed red cells should be planned when fetal or neonatal transfusion was anticipated. The association between previous transfusion and alloimmunization in this study reinforced the need for closer coordination between obstetric, transfusion-medicine and neonatal teams.

The clinical significance of maternal antibodies was demonstrated by the markedly poorer fetal and neonatal outcomes in antibody-positive pregnancies. Fetal anaemia occurred in 22.2% of antibody-positive pregnancies compared with 0.4% of antibody-negative pregnancies, while 11.1% required intrauterine transfusion. Preterm delivery and low birth weight were also significantly more frequent. These outcomes reflected both the direct effects of fetal haemolysis and the need for medically indicated early delivery in pregnancies at risk of progressive anaemia. Contemporary management relied on serial antibody assessment, paternal or fetal antigen determination where available, and middle cerebral artery peak systolic velocity surveillance. A value above approximately 1.5 multiples of the median was commonly used to identify fetuses at increased risk of moderate-to-severe anaemia and to guide fetal blood sampling or intrauterine transfusion. Saha et al. reported favourable outcomes using middle cerebral artery Doppler surveillance and intrauterine transfusion in RhD-alloimmunized pregnancies in India [16]. Indian studies of intrauterine transfusion have demonstrated that early diagnosis, appropriate timing and performance by an experienced fetal-medicine team can substantially improve perinatal survival [17]. More recent Indian experience has shown progressive improvement in outcomes over two decades through better Doppler surveillance, ultrasound-guided intravascular transfusion, blood-component preparation and neonatal support [18]. Nevertheless, hydrops, very early presentation, severe anaemia at the first transfusion and the requirement for multiple procedures remained adverse prognostic factors. The need for two intrauterine transfusions in the present cohort demonstrated that antibody screening was not merely a laboratory exercise but could identify pregnancies requiring life-saving fetal therapy.

Neonatal morbidity was also substantially greater among antibody-positive women. Positive direct antiglobulin tests occurred in half of exposed neonates, while 44.4% required phototherapy and 11.1% required exchange transfusion. Neonatal intensive care admission was approximately five times more frequent, and perinatal mortality was 11.1% compared with 1.0% among antibody-negative pregnancies. Maternal immunoglobulin G antibodies cross the placenta, coat antigen-positive

fetal erythrocytes and cause haemolysis, resulting in anaemia, hyperbilirubinaemia, hepatosplenomegaly, hydrops and, in severe cases, fetal or neonatal death. Even after a successful intrauterine transfusion and apparently stable delivery, neonates may develop delayed anaemia because maternal antibodies remain in the circulation and endogenous erythropoiesis may remain suppressed. Consequently, cord blood grouping, direct antiglobulin testing, haemoglobin, reticulocyte count and bilirubin estimation were essential, followed by serial monitoring after discharge when indicated. The present findings emphasized that antibody specificity, titre and obstetric history must be interpreted together; titre alone may not reliably predict severity for anti-K or in women who previously had a severely affected fetus. A major strength of this study was its prospective follow-up from antibody screening to obstetric and neonatal outcomes. However, its single-centre design and small number of antibody-positive pregnancies limited antibody-specific comparisons and multivariable modelling. Paternal phenotyping, fetal genotyping and long-term neonatal follow-up might not have been available in every case. Despite these limitations, the study provided strong evidence that universal antenatal antibody screening, careful transfusion history, timely anti-D prophylaxis, access to antibody identification and multidisciplinary fetal surveillance could reduce preventable fetal and neonatal morbidity. Larger multicentre Indian studies are required to establish region-specific screening schedules, critical titres and cost-effective strategies for extended antigen matching.

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